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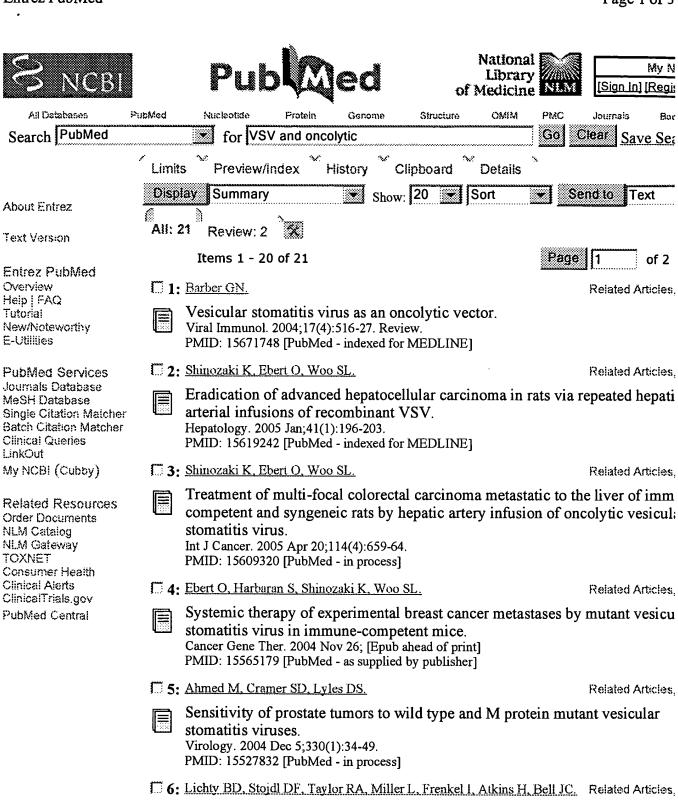
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	L24	L19 and balachadran S.in.	0
	L23	Woo S.in. and L19	0
	L22	L19 and Whitt M.in.	1
	L21	L19 and Connor J.in.	0
	L20	L19 and Ahmed M.in.	0
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	L18	L17 and oncolytic	0
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	L16	114 and oncolysis	0
	L15	L14 and oncolytic	0
	L14	Ebert O.in.	3
	L13	111 and oncolysis	0
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	L11	shinozaki K.in.	124
	L10	L9 and oncolytic	0
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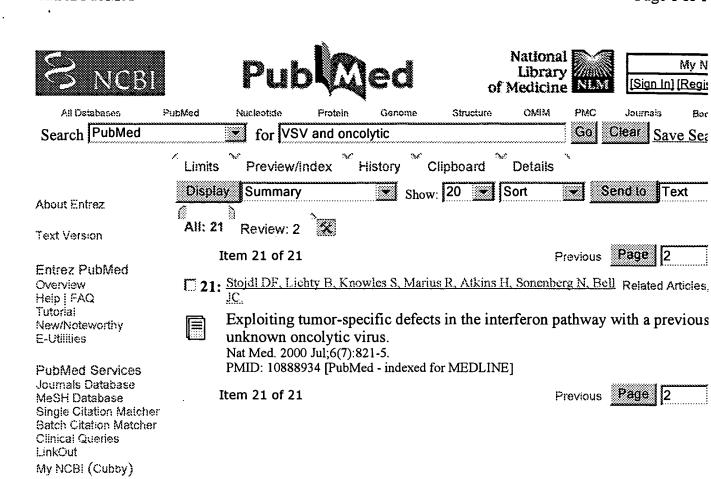
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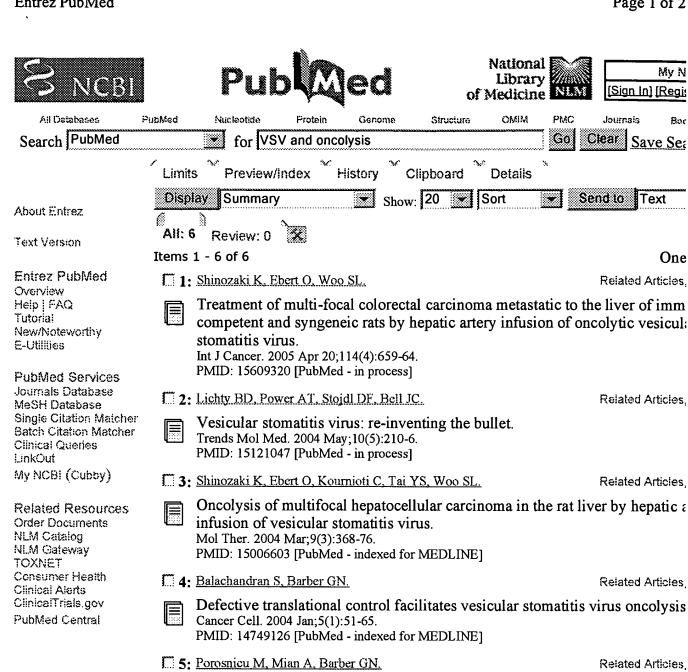
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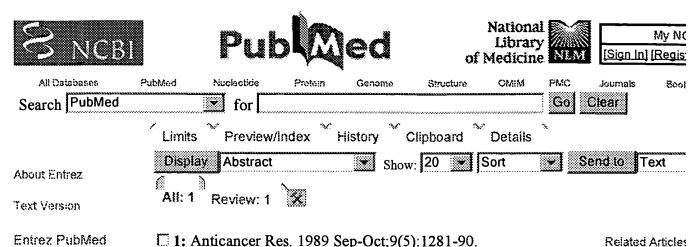
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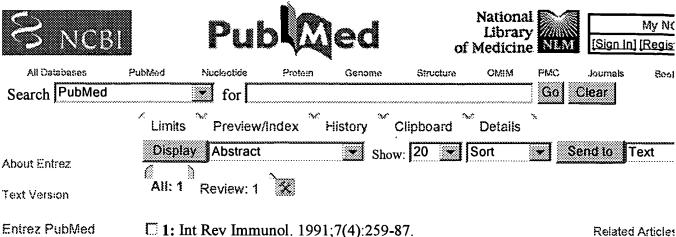
Department of Medicine, University of South Florida College of Medicine, Tampa.

Viruses can render services to mankind. 1. Retroviruses pinpoint and transdu cellular oncogenes. 2. Retroviral vectors can introduce antioncogenes (the RI gene) into malignant cells thus rendering the recipient cells nonmalignant. 3. Oncolytic viruses lyse tumor cells. 4. Parvoviruses replicate only in dividing and exert lysis and antioncogene effect in tumor cells without affecting restir. normal cells. 5. Myxo- and paramyxoviruses (and other viruses) upgrade the immunogenicity of cell surface antigens thus eliciting rejection type host immunity against these cells which is operational against not virus-infected c of the same type (post-oncolytic antitumor immunity). 6. Viruses or virally infected cells (including tumor cells) induce the production of lymphokines a cytokines (interferons, interleukins and tumor necrosis factor) and activate N cells and specific immune T cells cytotoxic to virus-infected cells (including tumor cells). 7. Measles virus may activate suppressor cells and both directly infecting lymphoma cells) and indirectly (by inducing molecular mediators o suppressor mononuclear cells inhibitory to the growth of neoplastic lymphoic hematopoietic cells) induce remissions of lympho- and hematopoietic malignancies. 8. Retroviral vectors deliver genes into tumor cells for encodin new surface antigens that render the tumor cells highly antigenic and more vulnerable to rejection type immune reactions of the host. Examples illustrate statement. Immunotherapy of tumors with active tumor-specific immunizatic after the induction of suppressor cells by fetal antigens and the elimination of proliferating suppressor clones by cyclophosphamide will again be proposed

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Sinkovics JG.

Cancer Institute, St. Joseph's Hospital, Tampa, Florida.

Postoncolytic immunity entails immune reactions acquired through an oncoly virus infection or through repeated immunizations with viral oncolysates (or virally modified tumor cell membranes) that are valid and operational also as virally not modified tumor cells of the same type. NK cells react to budding virions, induce target cell lysis primarily but not exclusively by the productic granzymes and pore-forming proteins and operate without direction from me cells. In contrast, immune T cells (including some TIL) are MHC-restricted, under the direction of memory cells and lyse target cells primarily but not exclusively by the release of lymphotoxin (TNF beta) causing programmed c death (apoptosis) through endonuclease activation and target cell DNA fragmentation. This author proposes that it is not NK, but the immune T cells mediate postoncolytic immunity. Oncogene amplification may protect immortalized tumor cells even when expressing peptide antigens through MF molecules against lymphotoxin-mediated apoptosis; but virally-infected tume cells releasing budding virions remain susceptible to NK cells. Highly immunogenic viral oncolysates should present both budding virions for NK (and processed viral and tumoral peptide antigens co-jointly for immune T cel

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